09/863,063

Teller 09/863,063

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=> d his 1

     (FILE 'MEDLINE, HCAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     10:00:55 ON 24 SEP 2002)
L53          24 DUP REM L52 (16 DUPLICATES REMOVED)

=> d que 153
L1            7 SEA FILE=REGISTRY AQSVPPGDIQTQPGTKIVFN.{0-}EWFQGDGMVRRKNLPIEYNP
                /SQSP
L2            1 SEA L1
L3          224 SEA GREENSTEIN D?/AU
L4        28961 SEA MILLER M?/AU
L5       125375 SEA NEMATODE#
L6      5274302 SEA INHIBIT?
L7        33271 SEA SEXUAL?(5A) MATUR?
L8        33084 SEA OOCYTE#(5A) MATUR?
L9       117489 SEA OVULAT?
L10          25 SEA GONAD? (5A) SHEATH#(5A) CONTRACT?
L12       38632 SEA CAENORHABDITIS
L13       23217 SEA ASCARIS
L14        1628 SEA ROUNDWORM#
L15        9055 SEA HETERODERA
L40     2726048 SEA ANTIBOD? OR IMMUNOGLOBULIN#
L41          76 SEA (L3 OR L4) AND (L5 OR (L12 OR L13 OR L14 OR L15 OR L16 OR
                L17 OR L18 OR L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR
                L26 OR L27 OR L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR
                L35))
L42           4 SEA L41 AND L6
L43          20 SEA (L5 OR (L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR
                L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26 OR L27 OR
                L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35)) AND
                (L6(5A)((L7 OR L8 OR L9 OR L10)))
L44           9 SEA (L5 OR (L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR
                L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26 OR L27 OR
                L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35)) AND
                ((L36(3A) PROTEIN#)(5A)(MUTATION# OR MUTANT# OR DEFECT#))
L45         392 SEA (L5 OR (L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR
                L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26 OR L27 OR
                L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35)) AND
                ((L37 OR L38 OR L39))
L46           4 SEA L45 AND ((L7 OR L8 OR L9 OR L10))
L47         107 SEA (L5 OR (L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR
                L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26 OR L27 OR
                L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35)) AND
                (L36(3A) PROTEIN#) AND (BIND? OR L40)
L48           5 SEA L47 AND L6
L49         360 SEA (L5 OR (L12 OR L13 OR L14 OR L15 OR L16 OR L17 OR L18 OR
                L19 OR L20 OR L21 OR L22 OR L23 OR L24 OR L25 OR L26 OR L27 OR
                L28 OR L29 OR L30 OR L31 OR L32 OR L33 OR L34 OR L35)) AND
                (MSP OR MAJOR(A) SPERM(A) PROTEIN#)
L50          83 SEA L49 AND (L40 OR BIND?)
L51           4 SEA L50 AND FEMALE?
L52          40 SEA L2 OR (L42 OR L43 OR L44) OR L46 OR L48 OR L51
L53          24 DUP REM L52 (16 DUPLICATES REMOVED)


=> d ibib abs l53 1-24

L53  ANSWER 1 OF 24  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
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Search completed by David Schreiber 308-4292

Teller 09/863,063

ACCESSION NUMBER: 2002:369385 BIOSIS
DOCUMENT NUMBER: PREV200200369385
TITLE: Serine/threonine phosphorylation signaling pathways regulate the C. elegans ClC anion channel CLH-3.
AUTHOR(S): Rutledge, Eric (1); Nehrke, Keith; Strange, Kevin
CORPORATE SOURCE: (1) Anesthesiology, Vanderbilt Univ. Sch. Med, 1161 21st Avenue South, Nashville, TN, 37232 USA
SOURCE: FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp. A799. http://www.fasebj.org/. print.
Meeting Info.: Annual Meeting of Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB CLH-3 is a ClC-2 Cl- channel ortholog expressed in C. elegans oocytes. The channel is activated by **oocyte** swelling and meiotic **maturation**. Exposing **oocytes** to metabolic **inhibitors** for 15-20 minutes or dialyzing oocytes with an ATP-free, metabolic inhibitor-containing pipette solution activates CLH-3 in the absence of oocyte swelling or cell cycle progression. Incubation of oocytes with 100 nM calyculin A, a PP1/PP2A phosphatase inhibitor, inhibits swelling- and maturation-induced current activation almost completely. However, incubation and intracellular dialysis of cells for 10 min with 1 muM okadaic acid, a more potent PP2A inhibitor, has no effect on CLH-3 activity. These and other experimental observations demonstrate that serine/threonine dephosphorylation events, possibly mediated by a PP1-type phosphatase, activate CLH-3. Yeast two-hybrid analyses using the CLH-3 C-terminus as bait identified a STE-20-like kinase, a putative kinase regulatory protein and two putative scaffolding/cytoskeletal proteins that interact with the channel. We are currently using reverse genetics and protein chemistry to assess the functional role of these proteins in CLH-3 regulation.

L53 ANSWER 2 OF 24 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2002:674741 SCISEARCH
THE GENUINE ARTICLE: 580KZ
TITLE: Ingestion of double-stranded RNA by preparasitic juvenile cyst **nematodes** leads to RNA interference
AUTHOR: Urwin P E (Reprint); Lilley C J; Atkinson H J
CORPORATE SOURCE: Univ Leeds, Ctr Plant Sci, Leeds LS2 9JT, W Yorkshire, England (Reprint)
COUNTRY OF AUTHOR: England
SOURCE: MOLECULAR PLANT-MICROBE INTERACTIONS, (AUG 2002) Vol. 15, No. 8, pp. 747-752.
Publisher: AMER PHYTOPATHOLOGICAL SOC, 3340 PILOT KNOB ROAD, ST PAUL, MN 55121 USA.
ISSN: 0894-0282.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 33
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB RNA interference is of value in determining gene function in many organisms. Plant parasitic **nematodes** are refractory to microinjection as a means of introducing RNA and do not show any oral uptake until they are within plants. We have used octopamine to stimulate uptake by preparasitic second stage juveniles of two cyst **nematodes**, **Heterodera** glycines and **Globodera** pallida. This new technique was used to facilitate uptake of double

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stranded RNA (dsRNA) together with fluoroscein isothiocyanate as a visual marker. Targeting cysteine proteinases did not reduce the number of parasites but caused a shift from the normal **female**/male ratio of 3:1 to 1:1 by 14 days postinfection (dpi). Exposure of H. glycines to dsRNA corresponding to a newly characterized protein with homology to C-type lectins did not affect sexual fate, but 41% fewer parasites were recovered from the plants. As expected, treatment with dsRNA corresponding to the **major sperm protein** (**MSP**) had no effect on either parasite development or sexual fate over 14 days. Northern analysis showed lower transcript abundance for the two targeted mRNAs that occur in J2, plus a later **inhibition** for **MSP** transcripts when males developed sperm at 15 dpi. These findings establish a procedure for RNAi of plant parasitic **nematodes**.

L53 ANSWER 3 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002208070 EMBASE
TITLE: The MEP-1 zinc-finger protein acts with MOG DEAH box proteins to control gene expression via the **fem-3 3'** untranslated region in **Caenorhabditis** elegans.
AUTHOR: Belfiore M.; Mathies L.D.; Pugnale P.; Moulder G.; Barstead R.; Kimble J.; Puoti A.
CORPORATE SOURCE: A. Puoti, Department of Zoology, University of Fribourg, Perolles, CH-1700, Switzerland. Alessandro.Puoti@unifr.ch
SOURCE: RNA, (2002) 8/6 (725-739).
Refs: 35
ISSN: 1355-8382 CODEN: RNARFU
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
021 Developmental Biology and Teratology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Cell fates in the **Caenorhabditis** elegans germline are regulated, at least in part, at the posttranscriptional level. For example, the switch from spermatogenesis to oogenesis in the hermaphrodite relies on posttranscriptional repression of the **fem-3** mRNA via its 3' untranslated region (UTR). Previous studies identified three DEAH box proteins, MOG-1, MOG-4, and MOG-5, that are critical for the **fem-3 3'** UTR control. Here we describe MEP-1, a zinc-finger protein that binds specifically to each of these three MOG proteins and that is required for repression by the **fem-3 3'** UTR in vivo. To investigate its in vivo function, we generated a mep-1 deletion mutant. The mep-1 null phenotype suggests a broad role for MEP-1 in C. elegans development, as it is associated with early larval arrest. In addition, mep-1 mutants can be defective in gonadogenesis and oocyte production when derived from a heterozygous mother. We suggest that MEP-1 acts together with the MOG proteins to repress **fem-3** mRNA and that it also functions in other pathways to control development more broadly.

L53 ANSWER 4 OF 24 MEDLINE
ACCESSION NUMBER: 2002078577 MEDLINE
DOCUMENT NUMBER: 21663716 PubMed ID: 11805057
TITLE: The Drosophila melanogaster seminal fluid protein Acp62F is a protease **inhibitor** that is toxic upon ectopic expression.
AUTHOR: Lung Oliver; Tram Uyen; Finnerty Casey M; Eipper-Mains Marcie A; Kalb John M; Wolfner Mariana F

Search completed by David Schreiber 308-4292

CORPORATE SOURCE: Department of Molecular Biology and Genetics, Cornell University, Ithaca, New York 14853, USA.
CONTRACT NUMBER: 1 F32 GM17673 (NIGMS)
SOURCE: GENETICS, (2002 Jan) 160 (1) 211-24.
Journal code: 0374636. ISSN: 0016-6731.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 20020128
Last Updated on STN: 20020511
Entered Medline: 20020510

AB Drosophila melanogaster seminal fluid **proteins** stimulate **sperm** storage and egg laying in the mated female but also cause a reduction in her life span. We report here that of eight Drosophila seminal fluid proteins (Acps) and one non-Acp tested, only Acp62F is toxic when ectopically expressed. Toxicity to preadult male or female Drosophila occurs upon one exposure, whereas multiple exposures are needed for toxicity to adult female flies. Of the Acp62F received by females during mating, approximately 10% enters the circulatory system while approximately 90% remains in the reproductive tract. We show that in the reproductive tract, Acp62F localizes to the lumen of the uterus and the female's sperm storage organs. Analysis of Acp62F's sequence, and biochemical assays, reveals that it encodes a trypsin **inhibitor** with sequence and structural similarities to extracellular serine protease **inhibitors** from the **nematode Ascaris**. In light of previous results demonstrating entry of Acp62F into the mated female's hemolymph, we propose that Acp62F is a candidate for a molecule to contribute to the Acp-dependent decrease in female life span. We propose that Acp62F's protease **inhibitor** activity exerts positive protective functions in the mated female's reproductive tract but that entry of a small amount of this protein into the female's hemolymph could contribute to the cost of mating.

L53 ANSWER 5 OF 24 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:868136 HCAPLUS
DOCUMENT NUMBER: 136:16722
TITLE: Identification of **nematode** control agents comprising **sexual maturation** and reproduction **inhibitors**
INVENTOR(S): **Greenstein, David; Miller, Michael A.**
PATENT ASSIGNEE(S): Vanderbilt University, USA
SOURCE: PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001089298	A2	20011129	WO 2001-US16452	20010521
WO 2001089298	A3	20020411		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,

Search completed by David Schreiber 308-4292

SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 2001071256 A5 20011203 AU 2001-71256 20010521
PRIORITY APPLN. INFO.: US 2000-205829P P 20000519
US 2001-274358P P 20010308
WO 2001-US16452 W 20010521

AB The present invention provides compns. and methods for identifying agents that stimulate or **inhibit nematode** reprodn., esp. related to **oocyte maturation**, sheath cell contraction, and **ovulation**. It is disclosed that the **major sperm protein** (**MSP**) is a signal for **female sexual maturation** in **nematodes** and provides compns. and methods for identifying anti-**nematode** agents with **MSP** as a target.

L53 ANSWER 6 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:245783 BIOSIS
DOCUMENT NUMBER: PREV200100245783
TITLE: Regulation and physiological role of a C. elegans ClC-2 ortholog.
AUTHOR(S): Rutledge, Eric (1); Christensen, Michael (1); Broslat, Adam (1); Bianchi, Laura (1); George, Alfred L. (1); Beld, Andrew (1); Morrison, Rebecca (1); **Greenstein, David (1)**; Strange, Kevin (1)
CORPORATE SOURCE: (1) Dept Anesthesiology, Cell Biology and Pharmacology, Vanderbilt Univ. Sch. Med., Nashville, TN, 37232 USA
SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A846. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB C. elegans oocytes express a swelling-activated Cl- chanel encoded by the ClC gene clh-3. The biophysical characteristics of CLH-3 are indistinguishable from mammalian ClC-2 and the channels share 40% amino acid identity. The strong conservation of biophysical and structural properties suggest that CLH-3 and ClC-2 may have similar physiological roles and regulatory mechanisms. Because ClC-2 is activated by cell swelling, it has been proposed to function in cell volume homeostasis, a housekeeping process conserved in widely divergent species. However, knockdown of CLH-3 activity by RNA interference (RNAi) has no effect on the rate or extent of oocyte regulatory volume decrease. Basic mechanisms of cell cycle control are conserved in all eukaryotic organisms. We observed that the volume sensitivity of CLH-3 varies dramatically as a function of oocyte growth and development. In full-grown oocytes undergoing meiotic maturation. CLH-3 is constitutively activated. These findings indicate that CLH-3 is controlled by cell cycle events and suggest that it may regulate cell cycle-associated physiological processes important for reproduction. To test this hypothesis, we quantified several reproduction events. Brood size and the timing of meiotic maturation, ovulation and fertilization were unaffected by RNAi of CLH-3. Meiotic maturation induces ovulatory contractions of gap junction-coupled gonadal sheath cells. Sheath contractions were initiated apprx2 min earlier in

Search completed by David Schreiber 308-4292

RNAi compared to control animals. These results demonstrate that CLH-3 modulates ovulation via oocyte-sheath cell intercellular signaling pathways. We propose that CLH-3 functions as a cell cycle sensor to ensure that **ovulation** is synchronized with meiotic maturation. **Inhibition** of sheath contractile activity by CLH-3 reduces the energetic costs of reproduction and may enhance reproductive success in C. elegans. We suggest that ClC-2 may be regulated by cell cycle events and function in intercellular communication pathways.

L53 ANSWER 7 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:244259 BIOSIS
DOCUMENT NUMBER: PREV200100244259
TITLE: Cell cycle progression activates a ClC-2 ortholog in C. elegans oocytes.
AUTHOR(S): Boehmer, Christoph (1); Rutledge, Eric (1); Miller, Michael (1); Greenstein, David (1); Strange, Kevin (1)
CORPORATE SOURCE: (1) Dept. Anesthesiology, Pharmacology and Cell Biology, Vanderbilt Univ. Sch. Med., Nashville, TN, 37232 USA
SOURCE: FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A846. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB C. elegans oocytes express an inwardly rectifying, swelling-activated Cl- channel with biophysical characteristics indistinguishable from heterologously expressed mammalian ClC-2. The oocyte channel is encoded by ClC gene clh-3. CLH-3 AND ClC-2 share 40% amino acid identity. The volume sensitivity of CLH-3 varies by >50-60 fold during oocyte growth and development. CLH-3 in small, early stage oocytes requires substantially more swelling to activate compared to larger, later stage oocytes. Upon completion of growth, **oocytes** undergo meiotic **maturation** and are **ovulated** and fertilized. In full-grown, **maturing oocytes**, CLH-3 is constitutively activated. Mean whole-cell. Cl- currents at -70 mV were -5.6 +- 1.5 pA/pF and -16.8 +- 3.3 pA/pF in non-maturing and full-grown, **maturing** wild type **oocytes** , respectively. These findings suggest that either completion of **oocyte** growth or induction of **maturation** trigger the channel. To begin addressing this issue, we are carrying out patch clamp studies on mutant worm strains where oocyte growth or cell progression are disrupted **fog-2** (feminization of germline) hermaphrodite worms possess a mutation that blocks sperm production. Sperm produce signals that induce meiotic **maturation**. Late stage **fog-2 oocytes** reach full-grown size, but **mature** only sporadically at rates <1/40th that of wild type animals. Whole-cell Cl- current in late stage, full-grown non-**maturing fog-2 oocytes** was - 3.2 +- 0.9 pA/pF. Meiotic **maturation** was induced in **fog-2 oocytes** by injecting into the gonad via the vulva recombinant Major Sperm Protein (MSP)38. CLH-3 was constitutively activated in late stage, **maturing oocytes** from MSP38-injected **fog-2** worms (Cl- current = - 39.7 +- 12.6 pA/pF). Taken together, these results indicate that CLH-3 is activated by induction of meiotic **maturation** rather than completion of **oocyte** growth. We suggest that signaling events associated with cell cycle progression regulate CLH-3.

Search completed by David Schreiber 308-4292

L53 ANSWER 8 OF 24 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2000:808608 SCISEARCH
THE GENUINE ARTICLE: 366NR
TITLE: CPEB proteins control two key steps in spermatogenesis in C-elegans
AUTHOR: Luitjens C; Gallegos M; Kraemer B; Kimble J; Wickens M (Reprint)
CORPORATE SOURCE: UNIV WISCONSIN, DEPT BIOCHEM, MADISON, WI 53706 (Reprint); UNIV WISCONSIN, DEPT BIOCHEM, MADISON, WI 53706; UNIV WISCONSIN, PROGRAM CELL & MOL BIOL, MADISON, WI 53706; UNIV WISCONSIN, HOWARD HUGHES MED INST, MADISON, WI 53706
COUNTRY OF AUTHOR: USA
SOURCE: GENES & DEVELOPMENT, (15 OCT 2000) Vol. 14, No. 20, pp. 2596-2609.
Publisher: COLD SPRING HARBOR LAB PRESS, 1 BUNGTOWN RD, PLAINVIEW, NY 11724.
ISSN: 0890-9369.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 44

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cytoplasmic polyadenylation element binding (CPEB) proteins bind to and regulate the translation of specific mRNAs. CPEBs from Xenopus, Drosophila, and Spisula participate in oogenesis. In this report, we examine the biological roles of all identifiable CPEB homologs in a single organism, **Caenorhabditis** elegans. We find four homologs in the C. elegans genome: cbp-1, cpb-**2**, cpb-**3**, and **fog-1**. Surprisingly, two homologs, CPB-**1** and **FOG-1**, have key functions in spermatogenesis and are dispensable for oogenesis. CPB-2 and CPB-3 also appear not to be required for oogenesis. CPB-1 is essential for progression through meiosis: cpb-1(RNAi) spermatocytes fail to undergo the meiotic cell divisions. CPB-1 protein is present in the germ line just prior to overt spermatogenesis; once sperm differentiation begins, CPB-1 disappears. CPB-1 physically interacts with EBF, another RNA-binding protein and 3' UTR regulator. In addition to its role in controlling the sperm/oocyte switch, we find that EBF also appears to be required for spermatogenesis, consistent with its interaction with CPEB. A second CPEB homolog, FOG-I, is required for specification of the sperm fate. The **fog-1** gene produces **fog-1**(L) and **fog-1**(S) transcripts. The **fog-1**(L) RNA is enriched in animals making sperm and is predicted to encode a larger protein; **fog-1**(S) RNA is enriched in animals making oocytes and is predicted to encode a smaller protein. The relative abundance of the two mRNAs is controlled temporally during germ-line development and by the sex determination pathway in a fashion that suggests that the **fog-1**(L) species encodes the active form. In sum, our results demonstrate that, in C. elegans, two CPEB proteins have distinct functions in the germ line, both in spermatogenesis: **FOG-1** specifies the sperm cell fate and CPB-1 executes that decision.

L53 ANSWER 9 OF 24 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000140286 EMBASE
TITLE: EMB-30: An APC4 homologue required for metaphase-to-anaphase transitions during meiosis and mitosis in **Caenorhabditis** elegans.
AUTHOR: Furuta T.; Tuck S.; Kirchner J.; Koch B.; Auty R.; Kitagawa

Search completed by David Schreiber 308-4292

R.; Rose A.M.; **Greenstein D.**
CORPORATE SOURCE: D. Greenstein, Department of Cell Biology, Vanderbilt Univ. School of Medicine, Nashville, TN 37232, United States. david.greenstein@mcmail.vanderbilt.edu
SOURCE: Molecular Biology of the Cell, (2000) 11/4 (1401-1419). Refs: 71
ISSN: 1059-1524 CODEN: MBCEEV
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Here we show that emb-30 is required for metaphase-to-anaphase transitions during meiosis and mitosis in **Caenorhabditis** elegans. Germline-specific emb-30 mutant alleles block the meiotic divisions. Mutant oocytes, fertilized by wild-type sperm, set up a meiotic spindle but do not progress to anaphase I. As a result, polar bodies are not produced, pronuclei fail to form, and cytokinesis does not occur. Severe-reduction-of-function emb-30 alleles (class I alleles) result in zygotic sterility and lead to germline and somatic defects that are consistent with an essential role in promoting the metaphase-to-anaphase transition during mitosis. Analysis of the vulval cell lineages in these emb-30(class I) mutant animals suggests that mitosis is lengthened and eventually arrested when maternally contributed emb-30 becomes limiting. By further reducing maternal emb-30 function contributed to class I mutant animals, we show that emb-30 is required for the metaphase-to- anaphase transition in many, if not all, cells. Metaphase arrest in emb-30 mutants is not due to activation of the spindle assembly checkpoint but rather reflects an essential emb-30 requirement for M-phase progression. A reduction in emb-30 activity can suppress the lethality and sterility caused by a null mutation in mdf-1, a component of the spindle assembly checkpoint machinery. This result suggests that delaying anaphase onset can bypass the spindle checkpoint requirement for normal development. Positional cloning established that emb-30 encodes the likely C. elegans orthologue of APC4/Lid1, a component of the anaphase-promoting complex/cyclosome, required for the metaphase-to-anaphase transition. Thus, the anaphase-promoting complex/cyclosome is likely to be required for all metaphase-to-anaphase transitions in a multicellular organism.

L53 ANSWER 10 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:155243 BIOSIS
DOCUMENT NUMBER: PREV200200155243
TITLE: MSP mutant A79K inhibits the localized filament assembly that drives leading edge protrusion in the amoeboid sperm of **Ascaris.**
AUTHOR(S): Griffin, Paul A. (1); Baker, Anne M.; Stewart, Murray; Roberts, Thomas M.
CORPORATE SOURCE: (1) Biology/Cell Biology, Florida State University, 334 BIO Unit I, Tallahassee, FL, 32306-4370 USA
SOURCE: Molecular Biology of the Cell, (Dec., 2000) Vol. 11, No. Supplement, pp. 377a. http://www.molbiolcell.org/. print.
Meeting Info.: 40th American Society for Cell Biology Annual Meeting San Francisco, CA, USA December 09-13, 2000
ISSN: 1059-1524.
DOCUMENT TYPE: Conference
LANGUAGE: English

L53 ANSWER 11 OF 24 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000410863 MEDLINE

Search completed by David Schreiber 308-4292

DOCUMENT NUMBER: 20403304 PubMed ID: 10949028
TITLE: ARK-1 inhibits EGFR signaling in C. elegans.
AUTHOR: Hopper N A; Lee J; Sternberg P W
CORPORATE SOURCE: MRC Laboratory of Molecular Biology, Cambridge, United Kingdom.. nah@mrc-lmb.cam.ac.uk
SOURCE: MOLECULAR CELL, (2000 Jul) 6 (1) 65-75.
Journal code: 9802571. ISSN: 1097-2765.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ271057
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000907
Last Updated on STN: 20000907
Entered Medline: 20000831

AB A screen for synthetic enhancers of sli-1 identified ark-1 (forAck-related tyrosine kinase), a novel inhibitor of let-23 EGFR signaling in C. elegans. An ark-1 mutation synergizes with mutations in other negative regulators of let-23, resulting in increased RAS signaling. Genetic analysis suggests that ARK-1 acts upstream of RAS and is dependent upon SEM-5. ARK-1 **inhibits** LET-23-mediated **ovulation**, a RAS-independent function. ARK-1 physically interacts with SEM-5 in the yeast two-hybrid assay. We find that sem-5 also has a negative function in let-23-mediated ovulation and suggest that this negative function is mediated by the recruitment of inhibitors such as ARK-1.

L53 ANSWER 12 OF 24 MEDLINE
ACCESSION NUMBER: 2000062179 MEDLINE
DOCUMENT NUMBER: 20062179 PubMed ID: 10597043
TITLE: elt-1, a gene encoding a **Caenorhabditis** elegans GATA transcription factor, is highly expressed in the germ lines with **msp** genes as the potential targets.
AUTHOR: Shim Y H
CORPORATE SOURCE: Department of Diagnostic Pathology, Asan Medical Center, Seoul, Korea.. yshim@www.amc.seoul.kr
SOURCE: MOLECULES AND CELLS, (1999 Oct 31) 9 (5) 535-41.
Journal code: 9610936. ISSN: 1016-8478.
PUB. COUNTRY: KOREA (SOUTH)
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000210

AB The **Caenorhabditis** elegans ELT-1 protein, a homolog of the vertebrate GATA transcription factor family, is a transcription activator that can recognize the GATA motif. We previously showed that the elt-1 mRNA was primarily expressed in C. elegans embryos. To examine whether the elt-1 mRNA in embryos is maternal, paternal or zygotic, Northern blot analysis was performed with RNA isolated from the C. elegans germ-line mutant strains, fem-2 (b245)lf, fem-3 (q20)gf, him-8 (e1489), and glp-4 (bn2). This analysis revealed that the high level of elt-1 mRNA in the C. elegans embryos resulted from either the maternal or the paternal transcription, rather than from the zygotic expression. These results further demonstrated that elt-1 was highly expressed in the germ-line of both sexes. To investigate the possible target genes for the ELT-1 protein in the germ line, the ELT-1 protein was expressed and tested for its

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binding specificity to the GATA motif that is present in the promoter region of the C. elegans **major sperm protein** genes. It was found that two conserved cis-elements, AGATCT and AGATAA, in the proximal promoter region of the **msp** -113 gene provided the best recognition site for ELT-1. Mutational analysis showed that the GATC core sequence was necessary for strong transactivation of the reporter gene, and that the combination of GATC and GATA motif resulted in a stronger transactivation by ELT-1 than either the duplicated GATC or GATA motif. These results suggest that the potential target for the ELT-1 protein in the germ-line may be one of the **major sperm protein** gene family.

L53 ANSWER 13 OF 24 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999102119 MEDLINE
DOCUMENT NUMBER: 99102119 PubMed ID: 9882501
TITLE: On the control of oocyte meiotic maturation and ovulation in **Caenorhabditis** elegans.
AUTHOR: McCarter J; Bartlett B; Dang T; Schedl T
CORPORATE SOURCE: Department of Genetics, Washington University School of Medicine, St. Louis, Missouri, 63110, USA.
SOURCE: DEVELOPMENTAL BIOLOGY, (1999 Jan 1) 205 (1) 111-28. Journal code: 0372762. ISSN: 0012-1606.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990205

AB Prior to fertilization, oocytes undergo meiotic maturation (cell cycle progression) and ovulation (expulsion from the ovary). To begin the study of these processes in **Caenorhabditis** elegans, we have defined a time line of germline and somatic events by video microscopy. As the oocyte matures, its nuclear envelope breaks down and its cell cortex rearranges. Immediately thereafter, the oocyte is ovulated by increasing contraction of the myoepithelial gonadal sheath and relaxation of the distal spermatheca. By systematically altering the germ cell contents of the hermaphrodite using mutant strains, we have uncovered evidence of four cell-cell interactions that regulate maturation and ovulation. (1) Both spermatids and spermatozoa induce oocyte maturation. In animals with a feminized germline, **maturation** is **inhibited** and **oocytes** arrest in diakinesis. The introduction of sperm by mating restores maturation. (2) Sperm also directly promote sheath contraction. In animals with a feminized or tumorous germline, contractions are infrequent, whereas in animals with a masculinized germline or with sperm introduced by mating, contractions are frequent. (3 and 4) The maturing oocyte both induces spermathecal dilation and modulates sheath contractions at ovulation; dilation of the distal spermatheca and sharp increases in sheath contraction rates are only observed in the presence of a maturing oocyte.

L53 ANSWER 14 OF 24 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1998:906724 SCISEARCH
THE GENUINE ARTICLE: 137GQ
TITLE: Polymerization-defective **mutants** of MSP, the key motility **protein** in the amoeboid **sperm** of **Ascaris**.

Search completed by David Schreiber 308-4292

Teller 09/863,063

AUTHOR: Griffin P A (Reprint); Stewart M; Roberts T M
CORPORATE SOURCE: FLORIDA STATE UNIV, DEPT BIOL SCI, TALLAHASSEE, FL 32306; MRC, MOL BIOL LAB, CAMBRIDGE CB2 2QH, ENGLAND
COUNTRY OF AUTHOR: USA; ENGLAND
SOURCE: MOLECULAR BIOLOGY OF THE CELL, (NOV 1998) Vol. 9, Supp. [S], pp. 814-814.
Publisher: AMER SOC CELL BIOLOGY, PUBL OFFICE, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 1059-1524.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L53 ANSWER 15 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:26013 BIOSIS
DOCUMENT NUMBER: PREV199900026013
TITLE: Polymerization-defective **mutants** of MSP, the key motility **protein** in the amoeboid **sperm** of **Ascaris**.
AUTHOR(S): Griffin, P. A. (1); Stewart, M.; Roberts, T. M. (1)
CORPORATE SOURCE: (1) Dep. Biological Sci., Fla. State Univ., Tallahassee, FL USA
SOURCE: Molecular Biology of the Cell, (Nov., 1998) Vol. 9, No. SUPPL., pp. 141A.
Meeting Info.: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998 American Society for Cell Biology
. ISSN: 1059-1524.
DOCUMENT TYPE: Conference
LANGUAGE: English

L53 ANSWER 16 OF 24 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 96:911069 SCISEARCH
THE GENUINE ARTICLE: VW700
TITLE: GLD-1, a cytoplasmic protein essential for oocyte differentiation, shows stage- and sex-specific expression during **Caenorhabditis** elegans germline development
AUTHOR: Jones A R (Reprint); Francis R; Schedl T
CORPORATE SOURCE: UNIV PENN, DEPT BIOL, PHILADELPHIA, PA 19104 (Reprint); WASHINGTON UNIV, SCH MED, DEPT GENET, ST LOUIS, MO 63110
COUNTRY OF AUTHOR: USA
SOURCE: DEVELOPMENTAL BIOLOGY, (25 NOV 1996) Vol. 180, No. 1, pp. 165-183.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0012-1606.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 60
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB GLD-1, a putative RNA **binding** protein, is essential for oocyte development in **Caenorhabditis** elegans. A gld-1 null mutation abolishes hermaphrodite oogenesis and confers a tumorous germline phenotype in which presumptive **female** germ cells exit the meiotic pathway and return to the mitotic cell cycle. Here we demonstrate that gld-1(null) germ lines express **female**-specific, but not

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male-specific, molecular markers, indicating that gld-1 acts downstream of sexual fate specification to regulate oocyte differentiation. Immunolocalization studies identify GLD-1 as a cytoplasmic germline protein that displays differential accumulation during germline development. first, germ cells that are in the mitotic cell cycle contain low levels of GLD-1 that likely reflect a nonessential gld-1 function (negative regulation of proliferation in the mitotic germ line) revealed in previous genetic studies. Second, entry of presumptive oocytes into the meiotic pathway is accompanied by a strong increase in GLD-1 expression/accumulation. GLD-1 levels are high through the pachytene stage but fall to background as germ cells exit pachytene and complete oogenesis. The meiotic prophase accumulation pattern is consistent with GLD-1's essential role in oocyte differentiation, which may be to repress the translation of a subset of maternal RNAs synthesized during early oogenesis until late oogenesis when GLD-1 is absent. (C) 1996 Academic Press, Inc.

L53 ANSWER 17 OF 24 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96048010 MEDLINE
DOCUMENT NUMBER: 96048010 PubMed ID: 7549905
TITLE: Cellular effects of olomoucine, an inhibitor of cyclin-dependent kinases.
AUTHOR: Abraham R T; Acquarone M; Andersen A; Asensi A; Belle R; Berger F; Bergounioux C; Brunn G; Buquet-Fagot C; Fagot D; +
CORPORATE SOURCE: Mayo Clinic, Department of Immunology, Rochester, MN 55905, USA.
SOURCE: BIOLOGY OF THE CELL, (1995) 83 (2-3) 105-20.
Journal code: 8108529. ISSN: 0248-4900.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511
ENTRY DATE: Entered STN: 19951227
Last Updated on STN: 19970203
Entered Medline: 19951114

AB Olomoucine (2-(2-hydroxyethylamino)-6-benzylamino-9-methylpurine) has been recently described as a competitive inhibitor (ATP-binding site) of the cell cycle regulating p34cdc2/cyclin B, p33cdk2/cyclin A and p33cdk2/cyclin E kinases, the brain p33cdk5/p35 kinase and the ERK1/MAP-kinase. The unusual specificity of this compound towards cell cycle regulating enzymes suggests that it could inhibit certain steps of the cell cycle. The cellular effects of olomoucine were investigated in a large variety of plant and animal models. This compound inhibits the G1/S transition of unicellular algae (dinoflagellate and diatom). It blocks Fucus zygote cleavage and development of Laminaria gametophytes. Stimulated Petunia mesophyl protoplasts are arrested in G1 by olomoucine. By arresting cleavage it blocks the Laminaria gametophytes. Stimulated Petunia mesophyl protoplasts are arrested in G1 by olomoucine. By arresting cleavage it blocks the development of Calanus copepod larvae. It reversibly inhibits the early cleavages of **Caenorhabditis** elegans embryos and those of ascidian embryos. Olomoucine inhibits the serotonin-induced prophase/metaphase transition of clam oocytes; furthermore, it triggers the the release of these oocytes from their meiotic metaphase I arrest, and induces nuclei reformation. Olomoucine slows down the prophase/metaphase transition in cleaving sea urchin embryos, but does not affect the duration of the metaphase/anaphase and anaphase/telophase transitions. It also inhibits the prophase/metaphase

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transition of starfish oocytes triggered by various agonists. Xenopus oocyte maturation, the in vivo and in vitro phosphorylation of elongation factor EF-1 are **inhibited** by olomoucine. Mouse **oocyte maturation** is delayed by this compound, whereas parthenogenetic release from metaphase II arrest is facilitated. Growth of a variety of human cell lines (rhabdomyosarcoma cell lines Rh1, Rh18, Rh28 and Rh30; MCF-7, KB-3-1 and their adriamycin-resistant counterparts; National Cancer Institute 60 human tumor cell lines comprising nine tumor types) is inhibited by olomoucine. Cell cycle parameter analysis of the non-small cell lung cancer cell line MR65 shows that olomoucine affects G1 and S phase transits. Olomoucine inhibits DNA synthesis in interleukin-2-stimulated T lymphocytes (CTLL-2 cells) and triggers a G1 arrest similar to interleukin-2 deprivation. Both cdc2 and cdk2 kinases (immunoprecipitated from nocodazole- and hydroxyurea-treated CTLL-2 cells, respectively) are inhibited by olomoucine. Both yeast and Drosophila embryos were insensitive to olomoucine. Taken together the results of this Noah's Ark approach show that olomoucine arrests cells both at the G1/S and the G2/M boundaries, consistent with the hypothesis of a prevalent effect on the cdk2 and cdc2 kinases, respectively.

L53 ANSWER 18 OF 24 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 92407040 MEDLINE
DOCUMENT NUMBER: 92407040 PubMed ID: 1527173
TITLE: **Mutation** of a putative **sperm** membrane **protein** in **Caenorhabditis** elegans prevents sperm differentiation but not its associated meiotic divisions.
AUTHOR: L'Hernault S W; Arduengo P M
CORPORATE SOURCE: Department of Biology, Emory University, Atlanta, Georgia 30322.
CONTRACT NUMBER: 1101-AG-9-2113 (NIA)
GM40697 (NIGMS)
SOURCE: JOURNAL OF CELL BIOLOGY, (1992 Oct) 119 (1) 55-68.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M74319; GENBANK-M74320; GENBANK-M74321; GENBANK-M74322; GENBANK-M74323; GENBANK-M74324; GENBANK-X66083; GENBANK-X66084; GENBANK-Z14066; GENBANK-Z14067
ENTRY MONTH: 199210
ENTRY DATE: Entered STN: 19921106
Last Updated on STN: 19950206
Entered Medline: 19921019

AB Spermatogenesis in the **nematode Caenorhabditis** elegans uses unusual organelles, called the fibrous body-membranous organelle (FB-MO) complexes, to prepackage and deliver macromolecules to spermatids during cytokinesis that accompanies the second meiotic division. Mutations in the spe-4 (spermatogenesis-defective) gene disrupt these organelles and prevent cytokinesis during spermatogenesis, but do not prevent completion of the meiotic nuclear divisions that normally accompany spermatid formation. We report an ultrastructural analysis of spe-4 mutant sperm where the normally close association of the FB's with the MO's and the double layered membrane surrounding the FB's are both defective. The internal membrane structure of the MO's is also disrupted in spe-4 mutant sperm. Although sperm morphogenesis in spe-4 mutants arrests prior to the formation of spermatids, meiosis can apparently be completed so that

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haploid nuclei reside in an arrested spermatocyte. We have cloned the spe-4 gene in order to understand its role during spermatogenesis and the molecular basis of how mutation of this gene disrupts this process. The spe-4 gene encodes an approximately 1.5-kb mRNA that is expressed during spermatogenesis, and the sequence of this gene suggests that it encodes an integral membrane protein. These data suggest that mutation of an integral membrane protein within FB-MO complexes disrupts morphogenesis and prevents formation of spermatids but does not affect completion of the meiotic nuclear divisions in C. elegans sperm.

L53 ANSWER 19 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1992:64776 BIOSIS
DOCUMENT NUMBER: BR42:28676
TITLE: SPERM DIFFERENTIATION IS UNCOUPLED FROM MEIOSIS BY **MUTATIONS** IN A PUTATIVE **SPERM** MEMBRANE **PROTEIN** IN **CAENORHABDITIS**-ELEGANS.
AUTHOR(S): L'HERNAULT S W; ARDUENGO P M
CORPORATE SOURCE: DEP. BIOL., EMORY UNIV., ATLANTA, GA. 30322.
SOURCE: ABSTRACTS OF PAPERS PRESENTED AT THE THIRTY-FIRST ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CELL BIOLOGY, BOSTON, MASSACHUSETTS, USA, DECEMBER 8-12, 1991. J CELL BIOL, (1991) 115 (3 PART 2), 48A.
CODEN: JCLBA3. ISSN: 0021-9525.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L53 ANSWER 20 OF 24 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 89089832 MEDLINE
DOCUMENT NUMBER: 89089832 PubMed ID: 3208296
TITLE: Relationship between plasma membrane mobility and substrate attachment in the crawling movement of spermatozoa from **Caenorhabditis** elegans.
AUTHOR: Pavalko F M; Roberts T M; Holliday L S
CORPORATE SOURCE: Department of Biological Science, Florida State University, Tallahassee.
CONTRACT NUMBER: GM-29994 (NIGMS)
SOURCE: CELL MOTILITY AND THE CYTOSKELETON, (1988) 11 (1) 16-23.
Journal code: 8605339. ISSN: 0886-1544.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198902
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19890221
AB **Caenorhabditis** elegans sperm are nonflagellated cells that lack actin and myosin yet can form pseudopods to propel themselves over solid substrates. Surface-attached probes such as latex beads, lectins, and antimembrane protein monoclonal **antibodies** move rearward over the dorsal pseudopod surface of sessile cells. Using monoclonal **antibodies** against membrane **proteins** of C. elegans **sperm** to examine the role of localized membrane assembly and rearward flow in crawling movement, we determined that substrates prepared by coating glass with antimembrane protein **antibodies**, but not naked glass or other nonmembrane-**binding proteins**, promote **sperm** motility. **Sperm** locomotion is **inhibited** in a concentration-dependent fashion when cells are

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bathed with soluble antimembrane protein monoclonal **antibodies** but not with antimouse Ig **antibodies** or a monoclonal **antibody** against a **sperm** cytoplasmic **protein**. Our results suggest that C. elegans sperm crawl by gaining traction with substrate-attached ligands via their surface proteins and by using the motor that moves those proteins rearward on unattached cells to pull the entire cell forward. Continuous insertion of new proteins at the front of the cell and their subsequent adhesion to the substrate allows this process to continue.

L53 ANSWER 21 OF 24 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1987:14675 HCAPLUS
DOCUMENT NUMBER: 106:14675
TITLE: Chitin synthetase inhibitors and their potential to control the root-knot **nematode**, **Meloidogyne** javanica
AUTHOR(S): Spiegel, Y.; Chet, I.
CORPORATE SOURCE: Dep. Nematol., ARO, Dagan, Israel
SOURCE: Nematologica (1985), 31(4), 480-2
CODEN: NEMAAT; ISSN: 0028-2596
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Rearing M. javanica on the roots of tomato cultured on agar spiked with Polyoxin D [22976-86-9] 100 or Dimilin [35367-38-5] 150 mg/L decreased the no. of eggs/g root to 17 and 26% of controls, resp. Alsystine, captan, thionex, Nikkomycine, and Trigard were ineffective. The 2 inhibitors of chitin synthetase [9030-18-6] may have acted by penetration of the vulval and anal outlets and direct action on the formation of eggs and their gelatinous envelope.

L53 ANSWER 22 OF 24 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1981:713 HCAPLUS
DOCUMENT NUMBER: 94:713
TITLE: Effect of tumor promoters on the **nematode**, **Caenorhabditis** elegans
AUTHOR(S): Miwa, Johji; Tabuse, Yo; Nishiwaki, Seiji; Furusawa, Mitsuru; Yamazaki, Hiroshi
CORPORATE SOURCE: Fac. Sci., Osaka City Univ., Osaka, Japan
SOURCE: Igaku no Ayumi (1980), 114(11), 910-12
CODEN: IGAYAY; ISSN: 0367-7826
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The addn. of 1-103 pg/mL doses of 12-O-tetradecanoylphorbol 13-acetate [16561-29-8], phorbol-12,13-didecanoate [24928-17-4], phorbol [17673-25-5], or 4.alpha.-phorbol [26241-63-4] to the culture medium of C. elegans **inhibited** growth, metamorphic development, and **ovulation**, depending on the promoter concns.

L53 ANSWER 23 OF 24 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1980:4693 BIOSIS
DOCUMENT NUMBER: BR18:4693
TITLE: EFFECTS OF GAMMA RADIATION ON THE ENTOMOGENOUS **NEMATODE** NEOAPLECTANA-CARPOCAPSAE.
AUTHOR(S): GAUGLER R; BOUSH G M
CORPORATE SOURCE: DEP. ENTOMOL., UNIV. WIS., MADISON, WIS. 53706, USA.
SOURCE: J. Invertebr. Pathol., (1979) 33 (1), 121-123.
CODEN: JIVPAZ. ISSN: 0022-2011.
DOCUMENT TYPE: Short Communication
FILE SEGMENT: BR; OLD

Search completed by David Schreiber 308-4292

LANGUAGE: English

L53 ANSWER 24 OF 24 MEDLINE
ACCESSION NUMBER: 77052740 MEDLINE
DOCUMENT NUMBER: 77052740 PubMed ID: 825598
TITLE: A comparison of immunologic asthma to two types of cholinergic respiratory responses in the rhesus monkey.
AUTHOR: **Miller M M**; Patterson R; Harris K E
SOURCE: JOURNAL OF LABORATORY AND CLINICAL MEDICINE, (1976 Dec) 88 (6) 995-1007.
Journal code: 0375375. ISSN: 0022-2143.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 197701
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19970203
Entered Medline: 19770125

AB Rhesus monkeys were used to characterize the respiratory response (RR) to aerosolized physostigmine (Phy) and compare the response to the acute, reagin-mediated RR and the carbachol response. In the latter two responses, there are characteristic increases in frequency of respiration (f), pulmonary resistance (PR), and decreases in peak expiratory flow rate (PEFR), tidal volume (TV), and dynamic compliance (C). In contrast, a Phy RR characteristically shows no change in TV and f. The Phy RR is **inhibited** by atropine and lidocaine. The carbachol RR is **inhibited** by atropine but not lidocaine. The Phy RR mimics a vagally induced RR more closely than carbachol because it is **inhibited** by pharmacologic and physiologic vagal blockade produced by atropine and lidocaine, respectively, whereas the carbachol RR is blocked by atropine but not lidocaine. The carbachol block by atropine is primarily not a block of vagal action, but a general pharmacologic block of cholinergic action. The reagin-mediated RR is not **inhibited** by pharmacologic or physiologic vagal blockade. We conclude that a Phy-induced vagomimetic RR differs from a reagin-mediated, immediate-type RR. Although a minor portion, at most, of a reagin-mediated, immediate-type RR may be mediated via the vagus nerve, the major portion of a reagin-mediated RR occurs independent of the influences of the parasympathetic innervation of the lung.

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